



NOTES

NEMATODES (ROUNDWORMS)

GENERALLY, WHAT ARE THEY?

PATHOLOGY & CAUSES

- AKA roundworms
 - *Microfilariae*: immature worms
 - *Microfilariae*: mature worms
- Parasites usually enter body cutaneously
 - Produce gastrointestinal (GI), occasionally cutaneous symptoms, disease

COMPLICATIONS

- Löffler syndrome
 - Transient pulmonary disease during select nematode development (*Necator Americanus*, *Strongyloides*, *Ascaris Lumbricoides*, *Toxocara*, *Ancylostoma duodenale*)
 - **Symptoms**: irritating, nonproductive cough, substernal burning discomfort, dyspnea, wheezing, fever, blood-tinged sputum production

SIGNS & SYMPTOMS

- GI/localized tissue symptoms, organism-dependent

DIAGNOSIS

LAB RESULTS

- Eosinophilia
- Gammaglobulinemia abnormalities (↑/↓ depending on organism)

Serology

- IgG levels
- Enzyme linked immunosorbent assays (ELISA), western blot

TREATMENT

MEDICATIONS

- Anthelmintic therapy
 - Nematode location-dependent

Intestinal nematodes

- Mebendazole/albendazole
 - Bind tubulin colchicine-sensitive sites → inhibit microtubule assembly → inhibited glucose uptake (without human effect) → organism death

Systemic nematodes

- Diethylcarbamazine (DEC)
 - Piperazine derivative (uncertain mechanism of action)
- Ivermectin
 - Synthetic lactone → negative-charged ion influx via glutamate-sensitive chloride channels → hyperpolarizes cells → helminth cell death
 - Not macrofilaricidal, pregnancy contraindication

VISIT...

LANZAROTE
Caliente.COM

ANCYLOSTOMA DUODENALE & NECATOR AMERICANUS

osms.it/ancylostoma-duodenale
osms.it/necator-americanus

PATHOLOGY & CAUSES

- Infectious **hookworm** species

Adult morphology

- *Ancylostoma duodenale*
 - Two ventral plates on buccal capsule's anterior margin; two large teeth fused at base (male 8–11mm, female 10–13mm long)
- *Necator americanus*
 - Two dorsal/ventral cutting plates around buccal capsule's anterior margin, paired subdorsal/subventral teeth (male 7–9mm, female 9–11mm long)
- *A. duodenale*, *Necator americanus*: most common hookworm species causing helminth gastrointestinal infection
 - Light infection (< 100 worms)
 - Moderate infection (100–500 worms)
 - Heavy infection (500–1000 worms)
- Pathophysiology
 - Hookworm attachment, hyaluronidase release → intestinal wall degradation, capillary laceration → anticoagulant peptides, inhibits platelet function inhibitor production → bleeding facilitation → extravasated blood ingested (approx. 0.5mL daily)
 - Moderate–heavy infections → chronic iron-deficiency anemia; protein malnutrition

Infectious form

- Filariform larva

Life cycle

- Infectious form maturation
 - Female *A. duodenale* lays 10,000–30,000 eggs daily

- Female *N. americanus* lays 5,000–10,000 eggs daily
- Human feces eggs release → soil deposition → eggs hatch (24–48 hours—favorable conditions) → rhabditiform larvae (L1) → transformation to infectious filariform larvae (L3) in 5–10 days
- Human stages
 - Filariform larvae skin penetration → passage through veins to heart, lung → pulmonary alveoli penetration, ascend bronchi to pharynx → coughed up/swallowed into small intestine → maturation → intestinal wall attachment via buccal capsule
- Some *A. duodenale* larvae undergo developmental arrest in gut tissues/muscle
 - Await more favorable environmental conditions (hypobiotic larvae)
- Natural life-span
 - *A. duodenale*: approx. one year
 - *N. americanus*: 3–5 years

Transmission

- Contaminated soil contact → percutaneous larval penetration
- Oral route
- Transmammary route

RISK FACTORS

- Inadequate clean-water access
- Poor sanitary conditions
- Walking barefoot (endemic areas)

COMPLICATIONS

- Severe iron-deficiency anemia; protein malnutrition → impaired growth, cognitive

development (children); heart failure (adults)

- Pregnancy
 - Low birth weight, maternal anemia, ↑ infant mortality

SIGNS & SYMPTOMS

- May be asymptomatic

Three phases

- Cutaneous phase
 - Local pruritic dermatitis (ground itch) with papular, sometimes vesicular, focal rash at larval penetration site (usually between toes)
- Pulmonary phase
 - Usually asymptomatic, may involve mild cough, pharyngeal irritation, sore throat, fever
- Gastrointestinal phase
 - Midepigastirc pain, appetite loss, nausea, diarrhea, vomiting

Heavy infection

- Hypoproteinemia → weight loss, anasarca, edema
- Anemia → fatigue, mental dullness, dyspnea, pallor
- Overt gastrointestinal bleeding

DIAGNOSIS

DIAGNOSTIC IMAGING

Abdominal X-ray

- Intestinal worm visualization

LAB RESULTS

- Acute infection
 - Eosinophilia
- Chronic infection
 - Anemia
- Kato-Katz method (thick smear)
- Polymerase chain reaction (PCR) test

Direct microscopy

- Stool specimen egg visualization

- *A. duodenale*, *N. americanus* eggs are morphologically indistinguishable

OTHER DIAGNOSTICS

- History
 - Contaminated-soil skin exposure; endemic-area travel; physical examination

TREATMENT

MEDICATIONS

- Anthelmintic treatment

ANGIOSTRONGYLUS (EOSINOPHILIC MENINGITIS)

osms.it/angiostrongylus

PATHOLOGY & CAUSES

- Parasitic nematode
 - Causes human GI/CNS disease
- *Angiostrongylus cantonensis*
 - Medically important species
 - Causes angiostrongyliasis (most common eosinophilic meningitis cause)

Adult morphology

- Three outer protective collagen layers; contains fully-developed gastrointestinal tract, simple stomal opening

Hosts

- *Primary intermediate host*: snails
- *Paratenic hosts*: fish, frogs, freshwater prawns
 - Not needed for developmental cycle
- *Definitive hosts*: wild rodents (brown, black rat)
- *Incidental hosts*: humans

Life cycle

- Infectious form maturation
 - Worms lay eggs in rat pulmonary artery → spread from lung capillaries to alveoli, larvae hatch → migrate up bronchi, trachea, across epiglottis → swallowed → fecal larvae passage → soil deposition → intermediate-host ingestion
- Human stages
 - Intermediate host larval ingestion (contaminated water/vegetable) → CNS tropism → migration into meningeal capillaries, brain tissue → meningoencephalitis
- Larvae usually fail to complete life-cycle, rarely → adult form in human host

Transmission

- Intermediate/paratenic host ingestion (raw/undercooked snails, fish, frogs), contaminated vegetables/water

Pathophysiology

- Post-inoculation, *A. cantonensis* larvae exhibit neurotropism → meninges, deeper brain tissue invasion → neural-tissue mechanical, toxic byproducts damage; antigen release → meningoencephalitis
- Migration to mesenteric arterioles → arteritis, thrombosis, small infarctions
 - May cause necrotic ulcers → peritonitis, fistula formation

COMPLICATIONS

- Long-term encephalitis → permanent nerve damage, intellectual disability, permanent brain damage, death

SIGNS & SYMPTOMS

- *Incubation period*: three weeks–two months
- *Meninges invasion*: meningitis picture
 - Severe headache, photophobia, stiff neck, fatigue, fever, hyperesthesia, vomiting, paresthesias
- *Brain parenchyma invasion*: encephalitis symptoms (brain location-dependent)
 - Cognitive impairment, slowed reactions, neuropathic pain, ascending weakness
 - t quadriparesis, areflexia, respiratory failure, muscle atrophy, death (rare)
- *Eye invasion*: visual impairment, pain, keratitis, retinal edema

DIAGNOSIS

LAB RESULTS

- Cerebrospinal fluid (CSF)
 - Eosinophilia
 - ↓ glucose levels (severe meningoencephalitis)

OTHER DIAGNOSTICS

- Endemic-area travel history
- Physical examination
- Clinical presentation

TREATMENT

- Self-limiting infection (usually)

MEDICATIONS

- Analgesics, sedatives
 - Treat headache, hyperesthesia
- Corticosteroids
- Anthelmintic therapy not advised
- Dying parasites, neurologic damage exacerbation → potential inflammatory response

OTHER INTERVENTIONS

- CSF drainage
 - Reduces intracranial pressure, relieve headache

ANISAKIS

osms.it/anisakis

PATHOLOGY & CAUSES

- Zoonotic roundworm
 - Causes human gastrointestinal, extra-gastrointestinal disease
 - Causative anisakiasis agent

Adult morphology

- Anteriorly-located mouth surrounded by projections; length (2cm/0.8in)

Infectious form

- L3 larvae

Hosts

- *Natural hosts*: marine mammals
- *Incidental hosts*: humans

Life cycle

- Infectious form maturation
 - Eggs hatch into larvae (seawater) → larval crustacean ingestion → fish/squid ingest crustaceans → muscular/subdermal larval encystation (L3

larvae) → marine mammals ingest fish → excystation, maturation, nematode reproduction → fecal egg release

- Human stages
 - Larval ingestion (infected fish) → larvae fail maturation, cannot complete life-cycle

Transmission

- Raw/undercooked fish ingestion

Pathophysiology

- Infected fish larvae ingestion → larvae burrow into intestinal wall, die → dead organism → inflammatory response → possible allergic reaction, abscess, mechanical obstruction
- If parasites pass into large intestine
 - Eosinophilic granulomatous response → mimic appendicitis/Crohn's disease

RISK FACTORS

- Raw/undercooked seafood ingestion (common in Japan)

COMPLICATIONS

- Small bowel obstruction
- Intestinal perforation
- Peritoneal cavity perforation (extraintestinal anisakiasis)
- Eosinophilic gastroenteritis/enterocolitis

SIGNS & SYMPTOMS

- Gastric anisakiasis
 - Acute epigastric pain, nausea, vomiting
- Intestinal anisakiasis
 - Severe abdominal pain, abdominal distension, palpable abdominal mass, intestinal obstruction, bloody diarrhea
- Allergic reactions
 - Urticaria, bronchoconstriction, angioedema, anaphylactic shock

DIAGNOSIS**DIAGNOSTIC IMAGING**

- Parasite visualization
 - Gastroscopic examination, emesis examination

LAB RESULTS

- ↑ serum IgE

TREATMENT**SURGERY**

- Parasite removal (endoscopically, surgically)

ASCARIS LUMBRICOIDES

osms.it/ascaris-lumbricoides

PATHOLOGY & CAUSES

- Intestinal roundworm parasite
 - Causative ascariasis agent
- Adult worm size: 15–30cm/5.9–11.8in
 - Largest intestinal nematode

Life cycle

- Female *A. lumbricoides* lays 200,000 eggs daily; begins egg-laying 9–11 weeks post-infection
- Infectious-form maturation
 - Eggs passed in stool → soil deposition → eggs embryonate; infectious after 2–4 weeks; can survive < ten years (favorable conditions)
- Human stages
 - Egg ingestion → larvae hatch → invade intestinal mucosa → portal circulation → systemic circulation → liver → lungs → larvae mature in alveoli (10–14 days) → ascend bronchial tree, pharynx, swallowed → larvae develop into adult

form in small intestine

- Life-span: 10–24 months

Pathophysiology

- Varies upon life-cycle stage
 - Pulmonary phase (early): caused by larval migration into lungs → pneumonitis
 - Intestinal phase: manifestations caused by adult-form presence → mechanical obstruction (degree worm-burden-dependent)

RISK FACTORS

- Egg-contaminated food/water ingestion (especially pig/chicken liver)
- Infected soil (children)
- Feco-oral route reinfection

COMPLICATIONS

- Intestinal obstruction
 - May → volvulus, ileocecal

intussusception, gangrene, intestinal perforation

- Children
 - Malnutrition/malabsorption, impaired growth, cognitive development
- Hepatobiliary involvement
 - Biliary colic, biliary strictures, obstructive jaundice, liver abscesses
- Pancreatic duct obstruction → pancreatitis
- Aspiration pneumonia during esophageal migration to trachea

SIGNS & SYMPTOMS

- Often asymptomatic
- **Pulmonary phase (Löffler syndrome):** develops 4–16 days post-infection
 - Dry cough, dyspnea, fever, wheezing, substernal discomfort, blood-tinged sputum; symptoms subside after 5–10 days
- **Intestinal phase:** develops 6–8 weeks post-infection
 - Abdominal discomfort, anorexia, nausea, vomiting, diarrhea

DIAGNOSIS

DIAGNOSTIC IMAGING

Chest X-ray

- Pulmonary phase
 - May show migratory bilateral oval infiltrates, range from several mm–several cm
- Intestinal phase
 - Adult *Ascaris* worm visualization, intestinal obstruction

CT scan

- Pulmonary phase
 - Multiple nodules, “ground-glass” attenuation
- Intestinal phase
 - “Bull’s eye” appearance (worm cross-section)

Barium swallow

- Intestinal phase
 - Defects in filling; if worms ingest contrast, tram-track appearance demonstrated

Microscopic examination

- Pulmonary phase
- Eosinophils, Charcot–Leyden crystals in sputum
- Intestinal phase
- Eggs/adult form **stool visualization** (Kato-Katz/FLOTAC method)

LAB RESULTS

- Pulmonary phase
 - Peripheral eosinophilia

OTHER DIAGNOSTICS

- Exposure/endemic-area travel history

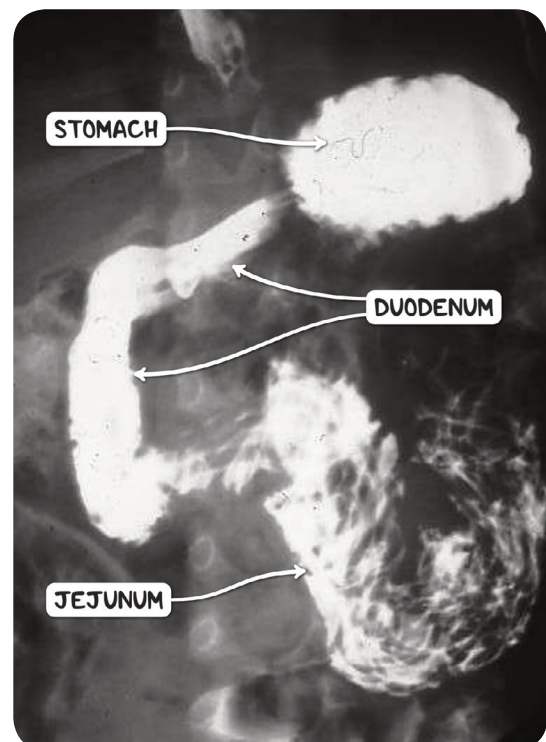


Figure 82.1 A barium study demonstrating ascariasis. There are numerous worms in the distal part of the duodenum and the ileum.

TREATMENT

MEDICATIONS

- Anthelmintic treatment
 - Only effective in intestinal phase
 - Pregnancy: pyrantel pamoate

SURGERY

- Complete intestinal obstruction, volvulus, intussusception, appendicitis, perforation cases

OTHER INTERVENTIONS

- Endoscopic retrograde cholangiopancreatography (ERCP)
 - Hepatobiliary-involvement cases

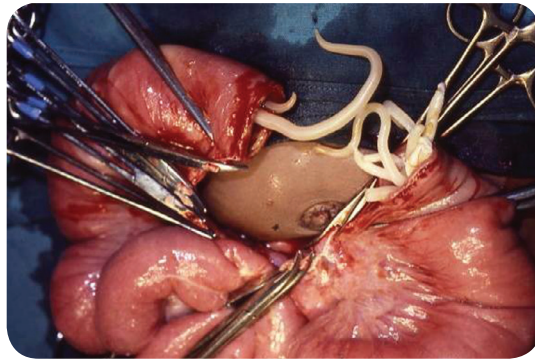


Figure 82.2 Ascaris worms emerging from the two free ends of the small intestine during an operation to remove a length of ischemic bowel.

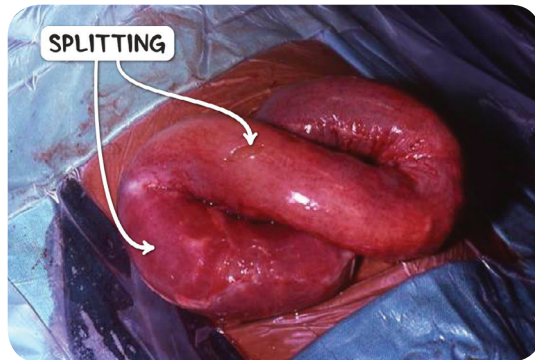


Figure 82.3 Small bowel packed with *Ascaris lumbricoides* worms. The bowel is distended so greatly that the visceral peritoneum on the antimesenteric border is split.

ENTEROBIUS VERMICULARIS (PINWORM)

osms.it/enterobius-vermicularis

PATHOLOGY & CAUSES

- Small roundworm
 - May infect human colon, rectum
 - Causative enterobiasis agent

Life cycle

- Female *E. vermicularis* lays > 10,000 eggs daily
- Perianal fold egg deposition → autoinfection by scratching; contaminated hand, mouth contact → eggs hatch into larvae (small intestine) → adult-form maturation (cecum, appendix)
- Life-span: 2–3 months

Transmission

- Scratching perianal area (autoinfection) → hand–mouth contact
- Contaminated hands (person–person)
- Contaminated surface contact
- Eggs may become airborne, inhaled

RISK FACTORS

- Crowded living conditions
- Children 5–10 years old

COMPLICATIONS

- Persistent perianal-area scratching, skin tearing → bacterial dermatitis, folliculitis
- Adult worms migrate to extraintestinal sites; cause vulvovaginitis, salpingitis, oophoritis, cervical granuloma, peritoneal inflammation

SIGNS & SYMPTOMS

- Mostly asymptomatic
- Perianal itching (*pruritus ani*) occurs nocturnally

- High worm burden → abdominal pain, nausea, vomiting

DIAGNOSIS

OTHER DIAGNOSTICS

- Pinworm paddle/Scotch tape test
 - Adhesive clear plastic paddle pressed against perianal areas → placed onto glass slide; reveals eggs upon microscopic examination

TREATMENT

MEDICATIONS

- Anthelmintic treatment

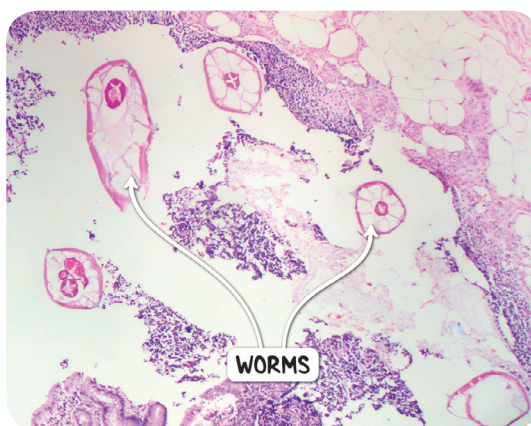


Figure 82.4 Pinworm found incidentally in an appendectomy specimen.

GUINEA WORM (DRACUNCULIASIS)

osms.it/guinea-worm

PATHOLOGY & CAUSES

- Water-borne nematode disease
 - **Characterization:** rash, GI illness with subcutaneous worm visualization

Life cycle/transmission

- Infected water → human copepod ingestion → GI, subcutaneous migration, sexual reproduction → pruritus, percutaneous larval eruption upon water-immersion → copepod larval consumption → two molting processes → pathogenic larvae in copepods

Pathogenesis

- GI symptoms
 - Ingested copepod death in GI system → larval migration into stomach, intestinal wall → entry into abdominal, retroperitoneal space
- Cutaneous symptoms
 - Larval sexual reproduction → female survival, skin migration → limb water-immersion → eruption

RISK FACTORS

- Rural Eastern-Africa residence/travel

COMPLICATIONS

- Ectopic site migration → abscess development
 - Lung, eye, pericardium, spinal cord
- Broken worm → intensely painful, edematous local, subcutaneous reaction

SIGNS & SYMPTOMS

- Systemic symptoms initially
 - Fever, urticaria, pruritus, dizziness, nausea/vomiting, diarrhea

- Cutaneous symptoms
 - Painful papule (2–7cm/0.8–2.8in) → enlarges, ↑ pain → worm emerges through ulceration

DIAGNOSIS

DIAGNOSTIC IMAGING

X-ray

- Calcified dead worm in subcutaneous tissue
 - If worm does not emerge through skin

OTHER DIAGNOSTICS

- Endemic-area travel/residence history
- Physical examination
 - Systemic → cutaneous symptom development

TREATMENT

OTHER INTERVENTIONS

- Worm extraction; careful extraction protocol
 - Multiple days (centimeters at a time); keeps worm intact, prevents local pruritic, edematous reaction



Figure 82.5 A match stick being used to extract a guinea worm from an ulcer on the leg.

Prevention

- Community surveillance, transmission methods education
- Safe water precautions
 - Nylon filters for water filtration, insecticides in drinking water courses, water source covering (prevents infected body-part immersion)
- Occlusive dressings applied to papules

LOA LOA (EYE WORM)

osms.it/loa-loa

PATHOLOGY & CAUSES

- Vector-borne filarial nematode endemic in Africa
 - **Characterization:** transient swelling episodes, subconjunctival adult worm migration
- AKA African eye worm
- Allergic reaction
- Subconjunctival infiltration
 - Associated with conjunctivitis, eyelid swelling

Life cycle/transmission

- Vector
 - Chrysops (biting deer fly; AKA tabanid fly)
 - Breed in rainforest canopies, lay eggs in muddy swamps; bite humans during daytime
- Tabanid fly human bite → filarial larvae transmission → three month maturation process → microfilarial production → bloodstream release (↑ release during daytime) → tabanid blood meal (infected individual) → filarial maturation in tabanid fly

RISK FACTORS

- Endemic-area residence/travel
 - Eastern, Central Africa

COMPLICATIONS

- Onchocerciasis co-infection
- Encephalitis (coincident headache, insomnia, coma may result)
- Cardiomyopathy (endomyocardial fibrosis)
- Nephropathy
- Arthritis
- Lymphadenitis
- Calabar swelling → entrapment neuropathy

SIGNS & SYMPTOMS

Allergic reactions

- Calabar swellings
 - 5–20cm/2–7.9in non-erythematous lesions
 - Transient, local subcutaneous swelling; face, extremities most common; localized pain, itching prior to swelling episodes
 - Urticaria, pruritus, asthma

Subconjunctival eye infection

- Commonly non-painful infiltration of eye subconjunctival area; conjunctivitis may occur (with eyelid swelling)
- Worm may be visible

DIAGNOSIS

LAB RESULTS

- Hypergammaglobulinemia
- ↑ IgE level
- Worm's presence (blood smear)
- Serology
 - IgG antibodies against *L. loa* antigens

OTHER DIAGNOSTICS

- History, physical examination
 - Worm detection in subcutaneous tissue, conjunctiva

TREATMENT

MEDICATIONS

- Diethylcarbamazine (DEC)
 - Active against *L. loa* microfilariae, microfilariae (adult worms)

- Onchocerciasis co-infection contraindicated (DEC provokes severe inflammatory skin, eye response; Mazzotti reaction)
- Eradication may require multiple treatment rounds
- Albendazole
 - Sterilizes mature worms without microfilarial activity
- Antihistamine/corticosteroids
 - Limits post-treatment immune reactions (e.g. Calabar swelling)

SURGERY

- Large worm removal

OTHER INTERVENTIONS

Prevention

- Weekly DEC prophylaxis
- Only considered for long-term endemic-area exposure

ONCHOCERCA VOLVULUS (RIVER BLINDNESS)

osms.it/onchocerca-volvulus

PATHOLOGY & CAUSES

- Filarial nematode transmitted by blackflies
- Leading preventable blindness cause in sub-Saharan Africa

Life cycle/transmission

- *Simulium* blackfly human bite → larvae skin deposited → adult parasite maturation (microfilariae) → subcutaneous/deeper intramuscular tissue migration → fibrous capsule/nodule development → reproduction → microfilarial (immature worm) production, subcutaneous tissue migration → human blackfly bite → microfilarial development into infective larvae (in blackfly)

Ocular onchocerciasis

- Common manifestation: West African savanna
- Commonly affects anterior eye chamber
 - Iridocyclitis, glaucoma, uveitis
- Posterior chamber may also be affected
 - Onchochorioretinitis, optic atrophy
- Ocular involvement degree correlates with symbiotic *Wolbachia* bacteria quantity

Onchocercal skin disease

- AKA Leopard/lizard/elephant skin (especially when depigmentation present); common manifestation in African forest areas
- Classified by chronicity
 - Acute/chronic papular onchodermatitis

- Lichenified onchodermatitis (AKA sowda/ black/dark); epidermal atrophy, elastic fiber breakdown may occur

PATHOLOGY

Subcutaneous involvement

- Onchocercoma
- Dermal *O. volvulus* → inflammatory response (prostaglandin E₂, transforming growth factor-beta-mediated) → nodule formation (onchocercoma); nodules predominate in bony prominence areas, peak inflammatory response occurs upon subcutaneous male *O. volvulus* death

Ocular involvement

- Anterior chamber disease
 - *O. volvulus* infiltration → immune response, *O. volvulus* death → Wolbachia release → innate immune response → corneal damage
- Posterior chamber disease
 - *O. volvulus* infiltration → immune response → cross-reactivity of *O. volvulus* antigen with retinal pigment epithelial protein → persistent immunologic response → inflammatory limbus, iris, choroid damage

RISK FACTORS

- West Africa, Eastern South America travel/ residence
 - Especially savanna, forest

SIGNS & SYMPTOMS

Cutaneous

- Pruritus
- Nodule development (lymphadenopathy may develop, persist depending on infection duration)
- Focal darkening/depigmentation
- Epidermal atrophy, hyperpigmentation, hyperkeratosis may be present

Ocular

- Punctate keratitis → fluffy corneal opacities → eosinophilic infiltrate → sclerosing keratitis → corneal opacification;

progressive (eventually irreversible) vision deficit

DIAGNOSIS

DIAGNOSTIC IMAGING

Ultrasound

- Deep onchoceroma detection

LAB RESULTS

- Eosinophilia
- Hypergammaglobulinemia
- PCR assay
 - *O. volvulus*

OTHER DIAGNOSTICS

- Rheumatologic/dermatologic onchocercoma evaluation

Skin snips

- Corneoscleral punch biopsy (ocular involvement)/ disposable razor blade for epidermal sample (cutaneous disease)
- 2+ snips taken in areas likely to harbor highest microfilariae concentration
- Positive only within 9–15 months post-infection (mature microfilariae produce microfilariae)

Patch test

- Topical DEC application → local skin reaction assessment
- Akin to Mazzotti reaction

Fundoscopy and corneal evaluation

- Slit-lamp examination reveals wriggling microfilariae
- Individuals sit forward for two minutes prior to examination (↑ microfilarial visualization likelihood on chamber examination)

TREATMENT

MEDICATIONS

- Ivermectin, doxycycline
 - **Ivermectin:** kills immature worms only, adult worm repopulate months after treatment

- **Doxycycline:** kills *Wolbachia* (symbiotic bacteria needed for *O. volvulus* fertility) for 24 months; block reproduction

OTHER INTERVENTIONS

Prevention

- Protective clothing, insect repellent (especially when blackflies most active—morning/evening)

STRONGYLOIDES STERCORALIS

osms.it/strongyloides-stercoralis

PATHOLOGY & CAUSES

- Filarial disease endemic in tropical areas with characteristic pulmonary infection route, complications include septic shock in immunocompromised individuals
- Mild GI, cutaneous, pulmonary inflammation, disease
 - Dermatitis, urticaria, duodenitis, enterocolitis, pneumonitis
 - May persist for years

Life cycle

- Larvae live in fecally-contaminated ground soil → enter human host through broken skin → hematologic spread → pulmonary alveolar sac infiltration, penetration → ascend tracheobronchial tree → swallowed → larval maturation in duodenum, jejunum → larval reproduction, fecal excretion
- **Autoinfection** (single-host life-cycle completion) may occur via larval perianal skin entry

PATHOLOGY

- Larvae contaminate skin through breakage → lung migration → inflammation infiltrate → intestinal wall migration → maturation, replication → larval intestinal mucosa penetration → autoinfection

RISK FACTORS

- Constipation, diverticula, steroid-use, ↓ bowel motility → ↑ autoinfection likelihood
- Hypogammaglobulinemia
- Anti-tumor necrosis factor receptor therapy
- Organ transplantation, immune suppression

COMPLICATIONS

- Hyperinfection (uncontrolled autoinfection → high worm-burden disease)
 - Immunocompromised individuals
 - Hematologic parasite spread includes CNS, heart, liver, lungs, endocrine glands
 - May manifest as septic shock

SIGNS & SYMPTOMS

- Immunocompetent individuals
 - Bloating, diarrhea
- Cutaneous
 - Edema, petechiae, serpiginous/urticarial tracking
- Gastrointestinal
 - Anorexia, nausea, vomiting, epigastric pain (duodenal ulceration cases), malabsorption (chronic enterocolitis)
- Pulmonary
 - Dry cough, throat irritation, dyspnea, wheezing, hemoptysis

DIAGNOSIS

DIAGNOSTIC IMAGING

Chest X-ray

- Pulmonary disease
 - Foci of hemorrhage, pneumonitis, pulmonary edema

LAB RESULTS

Organism identification

- Stool examination
 - Low sensitivity due to intermittent larval release
 - Repeat testing ↑ finding's reliability
- ELISA
 - Immunocompetent individuals

OTHER DIAGNOSTICS

- Endemic-area travel history

Endoscopy

- Histopathological (biopsy-driven) diagnosis
 - Stomach, duodenum, colon evaluation of mucosa appearance

TREATMENT

MEDICATIONS

- Ivermectin
- Albendazole
 - May combine with ivermectin in hyperinfection/disseminated disease states

OTHER INTERVENTIONS

Prevention

- Proper shoe wearing
 - Prevents broken-skin exposure
- Serologic evaluation
 - Solid organ transplant donors

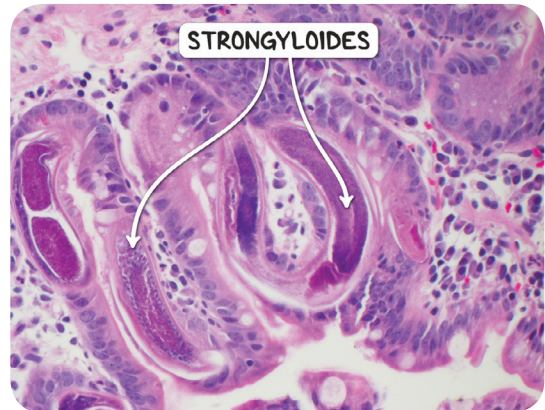


Figure 82.6 A duodenal biopsy containing an entire *Strongyloides* worm, likely an adult female.

TOXOCARA CANIS (VISCERAL LARVA MIGRANS)

osms.it/toxocara-canis

PATHOLOGY & CAUSES

- **Human roundworm disease:** dog, cat, pig vectors characterized by cutaneous/ocular disease
 - **Dogs:** *toxocara canis*
 - **Cats:** *toxocara cati*
 - **Pigs:** *Ascaris suum*

Life cycle/transmission

- **Definitive hosts:** cats, dogs, pigs
- **Accidental hosts:** humans
- Egg-laden stool → soil contamination → definitive host ingestion → GI tract reproduction → larval gut wall penetration → pulmonary migration, penetration → ascend tracheobronchial tree → swallowed → **small intestine worm maturation** → reproduction, egg excretion in stool
- **Paratenic host:** non-canine, small mammals; egg ingestion → larval gut wall penetration → tissue migration → cyst formation → human paratenic host ingestion → infection

Disease

- **Ocular involvement:** inflammatory granuloma of posterior pole, diffuse endophthalmitis, solitary peripheral retinal granuloma

RISK FACTORS

- Raw liver/undercooked meat ingestion (e.g. rabbit, chicken, cattle, pork)
- Children
 - Playground, sandbox use

COMPLICATIONS

- Hepatitis
- Pneumonitis

- Cardiac
 - Pericarditis, myocarditis
- **CNS involvement**
 - Myelitis, meningoencephalitis, seizure, cerebral vasculitis
- Ocular
 - Retinal detachment, **blindness**

SIGNS & SYMPTOMS

- **Visceral larva migrans**
 - Pruritic urticaria, fever, anorexia, malaise, irritability
- **Ocular larva migrans**
 - Unilateral **vision impairment**, uveitis, papillitis, endophthalmitis
- **Hepatomegaly**
- Respiratory distress

DIAGNOSIS

DIAGNOSTIC IMAGING

Chest X-ray

- Bilateral peribronchial infiltrates

CT scan

- **Chest:** multifocal subpleural nodules, "ground-glass" opacities, ill-defined margins
- **Abdomen:** multiple ill-defined lesions in liver parenchyma

Ultrasound

- **Abdomen:** may also identify lesions

LAB RESULTS

- Leukocytosis
- Hypogammaglobulinemia (↑ IgG, IgE)
- ELISA

- IgG antibodies against *Toxocara* excretory/secretory antigens
- Does not differentiate *Toxocara canis* from *cati*

OTHER DIAGNOSTICS

- Endemic-area travel history
- Physical examination

TREATMENT

MEDICATIONS

- Moderate → severe symptoms
 - Albendazole

- Severe inflammatory disease (myocarditis, pneumonitis, CNS involvement)
 - Prednisone
- Ocular larva migrans
 - Corticosteroids, albendazole

OTHER INTERVENTIONS

Prevention

- Hand hygiene practice
- Pet feces disposal, deworming
- Undercooked meat (especially liver) consumption risk education

TRICHINELLA SPIRALIS

osms.it/trichinella-spiralis

PATHOLOGY & CAUSES

- Roundworm infection (prevalent worldwide)
 - Raw/undercooked meat with encysted organism consumption
- *Trichinella*: nine species, twelve genotypes
 - Animal intermediate host-specific
 - *T. spiralis*: most common worldwide; variety of carnivorous, omnivorous animals

Life cycle/transmission

- Domestic cycle: involves pigs, rodents
- Sylvatic cycle: broad animal range
 - Bear, moose, wild boar most common human infection sources
- *Trichinella* larvae/mature worm ingestion by intermediate host → GI tract maturation → larvae release, migrate from female worms to striated muscles → encyst → intermediate host consumption (inadequate cooking temperature) → larval ingestion → larvae release in stomach (upon gastric acid, pepsin exposure) → small bowel mucosa invasion → adult worm maturation

→ female worm release larvae → striated muscle encystation

Disease

- Severity correlates with multiple factors
 - Number of ingested larvae directly correlates with ingested meat cooking temperature
 - *Trichinella* species
 - Incubation period (shorter correlates with ↑ disease severity)
- Gastrointestinal involvement
 - Maturing larvae burrow in intestinal mucosa
 - Diarrheal illness, nausea, vomiting
- Muscular involvement
 - Adult worm dissemination into skeletal muscle
 - Symptom resolution occurs with complete worm encystment
 - Mild subclinical fatigue, post-exercise weakness persists months–years

RISK FACTORS

- Undercooked/raw meat consumption

COMPLICATIONS

- Myositis, myocarditis
 - Eosinophil-enriched inflammatory response → life-threatening arrhythmia development
- Encephalitis, pneumonia
 - Direct larval parenchyma infiltration, respiratory muscle disease

SIGNS & SYMPTOMS

- Gastrointestinal
 - Nausea/vomiting, abdominal pain, diarrhea
- Muscular
 - Most severe symptoms
 - Pain (activity-dependent; ↑ muscle use/strain correlates with ↑ ↑ pain)
 - Tenderness, swelling, weakness
- Other
 - High fever, periorbital edema, chemosis, visual disturbance
 - Retinal hemorrhage on fundoscopic examination

DIAGNOSIS

DIAGNOSTIC IMAGING

CT scan/MRI

- In severe neurologic-involvement cases
- Multifocal cerebral cortex, white matter lesions

LAB RESULTS

- Serology
 - ELISA, western blot, immunofluorescence assays
 - Antibodies only detectable after 2-3 weeks of disease
 - Eosinophilia
 - Leukocytosis
 - ↑ Muscle enzymes, ↑ creatinine kinase, ↑ lactate dehydrogenase
- Muscle biopsy
 - Near tendinous insertion → highest yield obtained

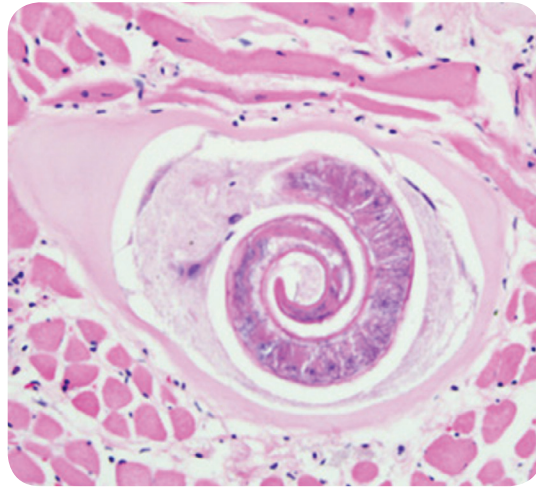


Figure 82.7 A muscle biopsy containing a *Trichinella* spp. larva.

TREATMENT

MEDICATIONS

- Mild disease
 - Self-resolving
 - Analgesia, antipyretics
- Systemic symptoms
 - Antiparasitic therapy (assists larval burrowing into intestinal mucosa treatment)
 - Corticosteroids

OTHER INTERVENTIONS

Prevention

- Postexposure prophylaxis in suspected cases
 - Mebendazole within six days of exposure
- Undercooked/raw meat consumption risk education, safe cooking practices

TRICHURIS TRICHIURA (WHIPWORM)

osms.it/trichuris-trichiura

PATHOLOGY & CAUSES

- Human intestinal parasite (AKA whipworm)
- *T. Trichiura* causative trichuriasis agent

Morphology

- Adult
 - 4 cm/1.6 in in length; made of anterior whip-like esophageal portion, posterior intestine/reproductive organ portion
- Egg
 - Prolate spheroids, polar plugs at each end

Life cycle

- Female *T. Trichiura* lays 3000–20,000 eggs daily 60–70 days post-infection
- Infectious form maturation
 - Unembryonated egg passage in stool → soil deposition → eggs embryonate, become infectious in 15–30 days
- Human stages
 - Egg ingestion → eggs hatch into larvae in small intestine → larvae mature into adult worms, embed in cecum, ascending colon
- Life-span: 1–3 years

RISK FACTORS

- Poor hygiene, sanitary conditions; inadequate human fecal disposal; uncooked/contaminated vegetable ingestion

COMPLICATIONS

- Heavy infection → rectal prolapse
 - Inflammation, edema caused by high embedded worm quantity in rectum (usually small children)
- Persistent blood loss → iron-deficiency anemia
- Children
 - Impaired growth, cognitive development

SIGNS & SYMPTOMS

- Mostly asymptomatic
- Heavier infection
 - Abdominal pain, distension, diarrhea, fecal blood/mucus, nocturnal stooling, tenesmus

DIAGNOSIS

OTHER DIAGNOSTICS

- Kato–Katz thick-smear technique
 - Eggs in stool visualization

TREATMENT

MEDICATIONS

- Anthelmintic treatment

WUCHERERIA BANCROFTI (LYMPHATIC FILARIASIS)

osms.it/wuchereria-bancrofti

PATHOLOGY & CAUSES

- Mosquito-borne nematode infection
 - **Characterization:** lymphatic, subcutaneous involvement → disfigurement, disability
- Mosquito species vector geographic-specific (e.g. *Culex*, *Aedes*)

Life cycle/transmission

- Filarial larvae introduction into human skin during mosquito bloodmeal → **lymphatic spread** → maturation into adult worm → reproduction, microfilarial hematologic release (commonly nocturnal release) → further transmission after infected individual's mosquito bite

Disease

- Adenolymphangitis, tropical pulmonary eosinophilia, hydrocele, chronic lymphatic disease

Pathogenesis

- Filarial antigens → T_H2 -type immune response → cytokine production (IL-5, IgE)
- Adult worm → mechanical lymphatic disruption → lymphangiectasia, lymphatic dilation
- Endosymbiotic bacteria *Wolbachia* → immune response potentiation

RISK FACTORS

- Sub-Saharan Africa, Southeast Africa, India, Pacific island travel/residence

COMPLICATIONS

- Lymphedema (AKA elephantiasis)
 - Chronic lymphatic vessel inflammation → limb swelling

- Chyluria
 - Intestinal lymphatic discharge into pelvis
 - May → nutritional deficiency (including anemia, hypoproteinemia)

SIGNS & SYMPTOMS

- Fever, hydrocele

Lymphatic disease

- Acute adenolymphangitis
- Painful lymphadenopathy, retrograde lymphangitis; self-resolving (4–7 days), may recur multiple times per year
- Dilation
- Lymphangiectasia
- Lymphedema
 - Pitting edema → non-pitting edema → limb hardening
 - Hyperpigmentation, hyperkeratosis may occur late in disease

DIAGNOSIS

DIAGNOSTIC IMAGING

Ultrasound

- Lymphatic dilation, lymphangiectasia, adult worms may be visualized

LAB RESULTS

- Circulating filarial antigen assays
 - **ELISA:** Og4C3 antigen detection
 - ↑ IgG4 levels indicate active infection
- Blood smear
 - Nocturnal blood draw important to ↑ microfilariae concentration

OTHER DIAGNOSTICS

- Physical examination
 - Wriggling adult filariae in lymphatic vessels

TREATMENT

MEDICATIONS

- DEC
 - **Contraindication:** onchocerciasis, severe Loa Loa infection, pregnancy
 - Doxycycline may be added as combination agent for direct larvicidal activity
 - ↓ lymphedema effectiveness
 - Proper skin care, topical antimicrobial administration (antifungal, antibacterial agents), extremity elevation, exercise may ↓ edema over time

SURGERY

- Hydrocele complications



Figure 82.8 A histological section of a lymph node which contains a filarial worm. The normal lymph node architecture has been replaced with an inflammatory infiltrate composed largely of eosinophils.

OTHER INTERVENTIONS

Prevention

- Mass drug administration programs
- Insecticide-treated bed nets
- Repellant, protective clothing in mosquito-endemic regions

NEMATODE DISEASE OVERVIEW

ORGANISM	TISSUE LOCATION (MACROFILARIA)	TISSUE LOCATION (MICROFILARIA)	KEY FACTS
A. DUODENALE, N. AMERICANIS	Small intestine	Connective tissue/blood	Vector/human source: fecally-contaminated water
ANGIOSTRONGYLUS	N/A	Central nervous system (CNS) capillaries	Vector/human source: under-cooked snails/slugs
ANISAKIS	N/A	Intestine	Vector/human source: under-cooked fish
A. LUMBRICOIDES	Intestine	Lung	Vector/human source: pig/chicken liver
E. VERMICULARIS	Cecum, appendix	Small intestine	Vector/human source: human-human
GUINEA WORM	Skin	Intestine	Vector/human source: copepods Careful extraction required
LOA LOA	Connective tissue	Blood	Vector/human source: deerfly (Chrysops) Calabar swelling, conjunctival infiltration
O. VOLVULUS	Connective tissue	Skin, eyes	Vector/human source: blackfly (Simulium) Causes blindness
S. STERCORALIS	Intestine	Connective tissue/blood	Vector/human source: fecally-contaminated water
T. CANIS	Intestine	Connective tissue/blood	Vector/human source: raw liver, under-cooked meat
T. SPIRALIS	Intestine	Striated muscle	Vector/human source: under-cooked meat Neurologic cysts possible
T. TRICHIURA	Cecum, appendix	Small intestine	Vector/human source: contaminated vegetables
W. BANCROFTI	Lymphatics	Blood	Vector/human source: mosquito Lymphedema; nocturnal periodicity